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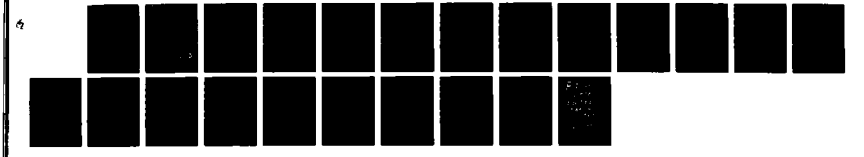

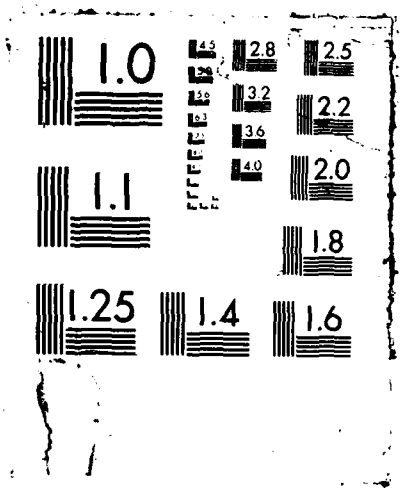
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Heat exchange through cutaneous vasodilation after atropine treatment in a cool environment

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ABSTRACT

This report summarizes a tightly controlled laboratory study in which the thermoregulatory effects of an intramuscular injection of atropine sulfate (2 mg) were compared with a placebo injection of sterile saline during exposure to a cool environment. Four subjects were tested during seated cycle exercise at a moderate exercise intensity (55% $\dot{V}_{O2}$ peak) at an ambient temperature of 22°C with an ambient water vapor pressure of 1.0 kPa. Esophageal temperature (T_{es}), mean weighted skin temperature ($\bar{T}_{sk}$), and forearm sweating rate ($\dot{m}_s$) were continuously measured during 30 minutes of rest and 35 minutes of exercise. Skin blood flow (FBF) from the forearm was measured twice each minute by venous occlusion plethysmography. Whole body sweating was calculated from weight changes pre- and post-exercise. The expected decrease in whole body and local sweating rate (-57% and -68%, respectively) occurred in the atropine treated subjects. By 10-15 min of exercise, radiative and convective heat exchange was significantly elevated from the head, chest, back, arm, forearm and thigh in the atropine experiments. Core temperature actually decreased 0.2°C in the atropine treated subjects during exercise as a result of enhanced dry heat exchange. By 25 min of exercise, FBF was 98% greater after atropine treatment. These results show that the peripheral modification of cutaneous blood flow which occurs in atropine treated subjects is sufficient to markedly alter heat exchange in a cool environment.

Key words: anticholinergic, dry heat exchange, sweating, thermoregulation, vasodilation

INTRODUCTION

The anticholinergic effect (6) of systemic atropine treatment on the eccrine sweat gland is well known and the inhibition of sweat secretion during exercise and heat stress is well documented in the scientific literature (1, 2, 3, 9, 10, 12, 15). An increased sweating rate in atropine treated male subjects has been shown following heat acclimation, combined with a greater dry heat exchange in these subjects after the 10 day acclimation period (9). Evidence for enhanced dry heat loss at ambient temperatures of 30°C (20% and 70% rh) and 35°C (50% rh) after atropine has been presented previously (10, 15). In addition, during low intensity exercise in severely hot (49°C, 20% rh) or hot (42°C, 20% rh) environments an increase in the skin temperature of atropine treated subjects decreased dry heat gain (9, 10).

The intramuscular injection of small doses of atropine sulfate (2 mg) is associated with widespread cutaneous vasodilation appearing thirty to forty-five minutes after treatment (12). We have reported a decreased dry heat gain in hot environments associated with this increased cutaneous vasodilation (9, 10). This cutaneous vasodilation is associated with a change in the central thermosensitivity of the forearm cutaneous blood flow to core temperature response during exercise in a warm environment (12) which may result from the activation of vasodilatory substances associated with the sweat gland.

These observations indicated that atropine altered the thermal gradient from the body surface to the environment and implied that excessive dry heat loss might occur in a cooler environment. In the current report, the cutaneous blood flow response was estimated by venous occlusion plethysmography, rather than calculated as changes in dry heat loss from the heat balance equation. In addition, heat exchange from

temperature gradients was calculated to further evaluate whether the regulation of body temperature in a cool environment may be compromised with the enhanced heat loss associated with atropine-induced cutaneous vasodilation.

METHODS

Four healthy male subjects were evaluated during seated cycle exercise in a cool environment (Table 1). All experimental procedures were identical with one exception; subjects exposed to 22°C (ambient water vapor pressure = 1.0 kPa) were studied on one occasion after 2 mg atropine sulfate (Elkins-Sinn, Cherry Hill, NJ) was injected into the vastis lateralis and once following a sterile saline placebo, injected in an identical manner. Subjects were not informed of the drug being injected and treatment order (drug or placebo) was counterbalanced. All procedures had been approved by the local human review committee.

The subject reported to the environmental chamber having not eaten for the previous twelve hours. He was weighed and then sat in a chair placed behind the pedals of a cycle ergometer, such that when pedalling his legs would be parallel to the floor. He swallowed an esophageal catheter containing a copper-constantan thermocouple and adjusted it to heart level for the measurement of esophageal temperature (T_{es}). Eight surface thermocouples (copper-constantan) were placed on the skin to estimate a mean weighted (13) skin temperature ($\bar{T}_{sk}$). Surface EKG electrodes were placed on the chest and back for the measurement of heart rate. Local sweating rate ($\dot{m}_s$) was measured from the left forearm with a small ventilated dew-point sensor (7, 11). Skin blood flow (FBF) was measured from the right forearm by venous occlusion plethysmography as described by Whitney (16) and modified by Hokanson (8).

At this point, the physician injected the atropine or the sterile saline. On-line data collection began. Metabolic heat production was evaluated from oxygen consumption measurements made during the 35 minute rest period and frequently during exercise. Exercise began at 55% of each subjects previously determined $\dot{V}_{O2}$ peak and continued for 30 minutes.

The maximal evaporative power of the environment was calculated as:

$$E_{max} = h_e(P_{s,sk} - P_{s,dp}) \qquad (W \cdot m^{-2})$$

where h_e is the evaporative coefficient and is equal to 13.2 $W \cdot m^{-2} \cdot K^{-1}$ in this study and $P_{s,sk}$ and $P_{s,dp}$ are the saturated water vapor pressures at the skin temperature and the ambient dew point temperature, respectively (4).

Evaporative heat loss from the skin surface was calculated from the weight loss during the experiment.

Dry heat loss was calculated as:

$$R+C = (h_r + h_c)(\bar{T}_{sk} - T_a) \qquad (W \cdot m^{-2})$$

where h_r and h_c are the radiative and convective coefficients and are equal to 6.0 and 4.7 $W \cdot m^{-2} \cdot K^{-1}$ respectively (13).

Net heat flow through the skin (M_{sk}) was calculated from the metabolic rate corrected for evaporative and convective heat exchange from respiration (5).

The data were analyzed by analysis of variance with repeated measures. All differences reported in the RESULTS are at $p<0.05$ unless otherwise noted.

RESULTS

The thermoregulatory and cardiovascular responses of the subjects at rest and during exercise are presented in Table 2. Heart rate was significantly increased during

rest and exercise in 22°C ($p<0.05$) after atropine administration. During exercise in the atropine treated subjects, sweating was significantly depressed ($p<0.05$) and forearm blood flow was enhanced ($p<0.05$). Specifically, whole body sweating was decreased an average of 57% and local (forearm) sweating was depressed an average of 68%. Esophageal temperature actually decreased during exercise (0.2°C) in the atropine experiment resulting from the increased dry heat loss. This response is shown in Figure 1 as the mean $\pm$ SD esophageal temperature response graphed over time in both control and atropine experiments. T_{es} leveled off at approximately 10 min of exercise in the control experiments, however in the atropine experiments T_{es} declines after this point. The metabolic rate was unchanged during this decrease in esophageal temperature (~700 W or 390 $W \cdot m^{-2}$), thus the decrease was the result of the increased heat loss. Forearm blood flow continued to increase throughout the exercise bout after atropine administration, but during the control experiment, FBF stabilized during steady-state exercise. This response is shown in Figure 2 as the mean $\pm$ SD forearm blood flow response graphed over time in both control and atropine experiments. The 98% increase in forearm blood flow seen during steady-state exercise in the atropine experiments (Table 2) resulted in a significantly higher $\bar{T}_{sk}$ (1.55°C, $p<0.05$).

The mean local skin temperatures at five minute intervals during rest and exercise is presented in Table 3 during control and atropine experiments. Seven of the eight skin sites measured show an increase in the atropine experiments, the calf being the exception. The mean local R+C is presented in Table 4 for control and atropine experiments. Between 10 and 15 min of exercise, R+C was significantly elevated from the head, chest, back, arm, forearm and thigh in the atropine experiments. Dry heat loss from the hand was higher in the atropine experiments after 25 minutes of exercise.

Dry heat loss from the calf (moving during exercise) was not different between the control and atropine experiments. A presentation of various calculated variables regarding heat exchange is shown in Table 5. These data indicate the altered heat exchange in the atropine experiments as E_{sk} was decreased -58% and dry heat exchange was increased approximately 15% at 22 to 27 minutes of exercise.

The onset time for forearm sweating was delayed in the atropine (11.5 min) experiments by an average of 6 minutes compared to the placebo experiments (5.5 min). Conversely, the onset of cutaneous blood flow occured 9 minutes earlier in atropine experiments (8.3 min).

DISCUSSION

The systemic administration of atropine sulfate in a volume equal to that contained in one field applicable auto-injector was sufficient to alter heat exchange in soldiers performing moderate exercise in a cool environment. The self-administration of this anti-cholinergic agent without exposure to a nerve agent challenge may occur in field situations due to fear or confusion. A series of studies at various levels of environmental stress or thermoregulatory strain have been conducted at our laboratory to evaluate the effectiveness of a soldier's performance following this inappropriate or accidental atropine administration (9, 10, 12, 15). The current study has extended our observations of the effects of atropine on thermoregulation by including the direct assessment of the vasomotor and local sudomotor responses to the whole body sweating responses during exercise in a cool environment. The increased blood flow to the skin surface increased the surface temperature and widened the thermal gradient between the skin and the ambient air providing for greatly enhanced dry heat loss from the subjects. The cutaneous vasodilation and the consonant dry heat loss seen in atropine treated subjects, in the absence of an anticholinergic nerve agent exposure,

was sufficient to decrease body temperature. In fact, this increased cutaneous perfusion actually caused core temperature to decrease by 0.2°C after T_{es} had stabilized at approximately 10 minutes of exercise. Further experiments will have to be done to more fully evaluate the implications of this increased convective and radiative heat loss in cold environments.

Heat loss through the hands must be investigated further as the control of hand blood flow is different from that of the forearm in that vasodilation is achieved through the release of vasoconstrictor activity, not through any vasodilatory mechanism (14). The temperature of the hand increased in the present study approximately 10 minutes following the increase in skin temperature of the other sites measured. In addition, subjects in the present study and our earlier studies (9, 10) reported hand swelling during exercise. The extent to which heat loss through the hand and/or swelling of the hand interferes with dexterity cannot be addressed from the current investigation. Furthermore, the possibility of increased risk of cold injury of the hands and possibly the feet in individuals treated with atropine during exposure to a more severe cold stress cannot be addressed from the data presented here.

It is important to note that the subjects in the present experiment were clothed in running shorts, shoes and socks to enable the appropriate measurements of heat exchange properties to occur. This is not the clothing that a soldier would wear in the field. However, in on-going studies at a very low exercise intensity, core temperature decreased over time in soldiers dressed in either the temperate battle dress uniform (BDU) or mission oriented protective posture IV (MOPP IV) configurations at both 12°C (MOPP IV) and 22°C (BDU) after systemic atropine administration.

The effect(s) that atropine has on heat exchange in hot environments has been thoroughly studied. However, associated problems during exposure to cold

environments have not been elucidated. The data from the present study indicate that even during moderate exercise in a cool environment (22°C, 72°F), enhanced dry heat exchange occurs. The altered heat exchange properties of individuals treated with atropine during exposure to more severe environments may increase the risk of cold injury in these individuals.

In summary, the anticipated decreased sweating rate and increased heart rate occurred with the systemic administration of 2 mg of atropine sulfate. Atropine caused widespread cutaneous vasodilation in healthy male subjects during moderate exercise in a cool environment which was manifested in enhanced dry heat exchange in this environment. During exercise in a cool environment, the increased cutaneous vasodilation is sufficient to lower the body temperature.

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The views, opinions, and/or findings contained in this report are those of the authors and should not be contrued as an official Department of the Army position, policy or decision unless so designated by other official documentation.

Human subjects participated in these studies after giving their informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

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Table 1. Subject characteristics.

	Age (yr)	Ht (cm)	Wt (kg)	SA[1] (m^2)	$\dot{V}O_2$ peak[2] ($l \cdot min^{-1}$)	Body fat[3] (%)
1	19	180.3	77.1	2.00	3.83	9.7
2	24	172.7	78.9	1.93	3.81	17.0
3	18	188.0	64.0	1.87	3.29	13.5
4	18	182.9	66.5	1.87	3.76	13.4
$\bar{X}$	20.0	181.6	71.6	1.92	3.67	13.4
sd	3.0	6.0	7.5	0.06	0.26	3.0

[1]DuBois body surface area.
[2]Measured while seated behind the cycle ergometer.
[3]Hydrostatic weighing.
The numbers 1 through 4 are the subject numbers.

Table 2. Mean ± SD temperature, forearm blood flow, local and whole body sweating measurements at rest and during exercise (22 to 27 min).

	T_{es} (°C)	$\bar{T}_{sk}$ (°C)	FBF (ml•100ml^{-1}•min^{-1})	$\dot{m}_s$ (mg•cm^{-2}•min^{-1})	HR (bpm)	$\dot{M}_s$ (g•min^{-1})
REST						
Saline	36.79 (0.20)	30.29 (0.51)	0.4 (0.1)	0.07 (0.01)	69 (18)	---
Atropine	36.81 (0.15)	30.14 (0.31)	0.4 (0.1)	0.07 (0.01)	86* (18)	---
EXERCISE						
Saline	37.36 (0.10)	31.01 (0.33)	4.1 (3.3)	0.77 (0.23)	138 (16)	9.4 (3.3)
Atropine	37.26 (0.22)	32.56* (0.87)	8.1* (3.6)	0.25* (0.17)	162* (13)	4.0* (2.7)

*$p<0.05$, different from saline. —
T_{es}, esophageal temperature; $\bar{T}_{sk}$, mean skin temperature; FBF, forearm blood flow; $\dot{m}_s$, forearm sweating; HR, heart rate; $\dot{M}_s$, whole body sweating rate.

Table 3. Local and mean skin temperatures (^{o}C) at five minute intervals during rest and exercise in both control and atropine experiments.

TIME (min)	Head	Chest	Back	Arm	Forearm	Hand	Thigh	Calf	Mean[1]
CONTROL									
-5	31.5	30.6	33.3	29.9	29.3	28.2	29.7	29.0	30.38
0	31.8	30.7	33.2	29.8	29.0	28.1	29.2	28.5	30.18
5	31.9	30.9	33.0	29.6	28.1	28.0	28.9	29.0	30.15
10	31.5	30.8	32.8	29.8	27.5	28.4	29.1	29.3	30.16
15	32.3	30.9	33.3	30.4	28.4	29.2	29.9	29.8	30.71
20	32.5	31.3	33.5	31.2	28.5	30.3	31.2	30.0	31.21
25	32.8	31.0	33.4	31.7	28.5	30.1	31.1	30.3	31.25
30	33.4	31.1	33.4	32.1	28.7	31.4	31.2	30.2	31.42
ATROPINE									
-5	32.5	30.0	32.9	30.0	29.1	26.6	29.6	28.8	30.13
0	32.5	29.9	33.0	29.8	28.8	26.5	29.4	28.3	29.96
5	32.7	30.0	32.8	29.6	28.6	26.3	29.0	28.3	29.84
10	33.7	31.0	33.2	29.8	28.4	28.2	29.9	29.2	30.60
15	34.5	32.4	34.1	31.5	29.3	29.0	31.5	29.6	31.66
20	34.7	32.9	34.7	33.3	30.5	30.5	32.9	29.4	32.38
25	34.7	33.0	34.9	34.4	31.3	31.8	33.6	30.2	32.92
30	34.7	33.2	35.3	35.0	32.4	33.2	34.1	30.5	32.85

[1]The mean skin temperature is calculated from an area weighting of the eight skin sites measured (13).

Table 4. Local[1] and mean dry heat exchange (R+C. $W \cdot m^{-2}$) at five minute intervals during rest and exercise in both control and atropine experiments.

TIME (min)	Head	Chest	Back	Arm	Forearm	Hand	Thigh	Calf	Mean[2]
CONTROL									
-5	102	92	121	85	78	66	82	75	90
0	105	93	120	83	75	65	77	70	87
5	106	95	118	81	65	64	74	75	87
10	102	94	116	83	59	68	76	78	87
15	110	95	121	90	68	77	85	83	93
20	112	96	123	98	70	88	98	86	99
25	116	96	122	104	70	87	97	89	99
30	122	96	122	108	72	101	98	88	101
ATROPINE									
-5	112	86	117	86	76	49	81	73	87
0	112	85	118	83	72	48	79	67	85
5	114	86	116	81	71	46	75	67	84
10	125	96	120	83	68	66	85	77	92
15	134	111	129	102	78	75	102	81	103
20	136	117	136	121	91	91	117	79	111
25	136	118	138	133	100	105	124	88	117
30	136	120	142	139	111	120	129	91	116

[1]These values are expressed in $W \cdot m^{-2}$ and have not been corrected for the area that the specific segment contributes to the entire body surface area.

[2]The mean R+C was calculated from the mean skin temperature and is equivilent to a mean based on an area weighting of the eight skin sites (13).

Table 5. Calculated parameters for the assessment of heat exchange during exercise (22-27 min) for the four subjects in control (saline) and atropine experiments.

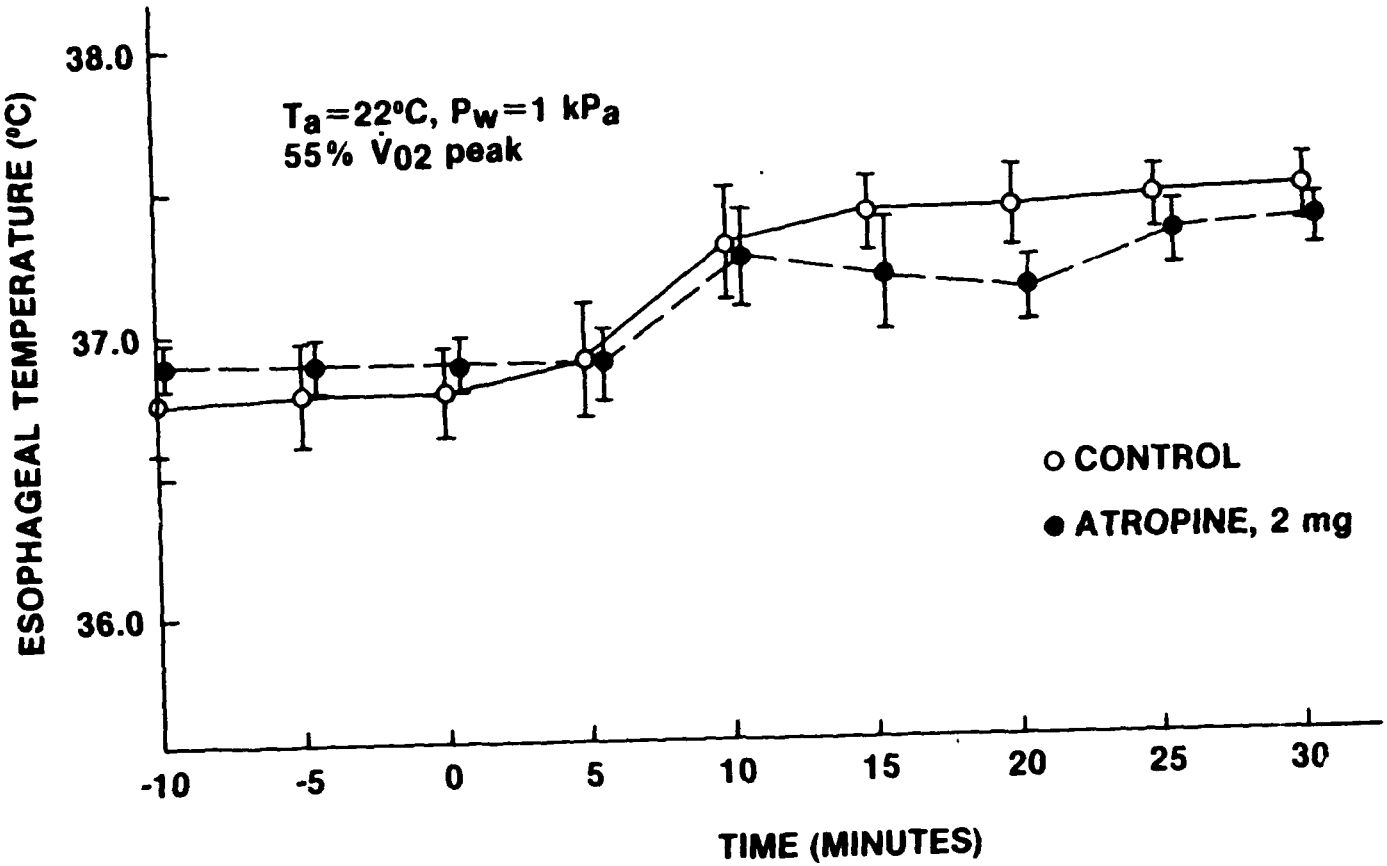
	E_{max} ($W \cdot m^{-2}$)	E_{sk} ($W \cdot m^{-2}$)	R+C ($W \cdot m^{-2}$)	M_{sk} ($W \cdot m^{-2}$)	$P_{s,sk}$ (kPa)	w (%)
Saline	353	198	96	346	4.5	.56
Atropine	392	84	113	347	4.9	.21

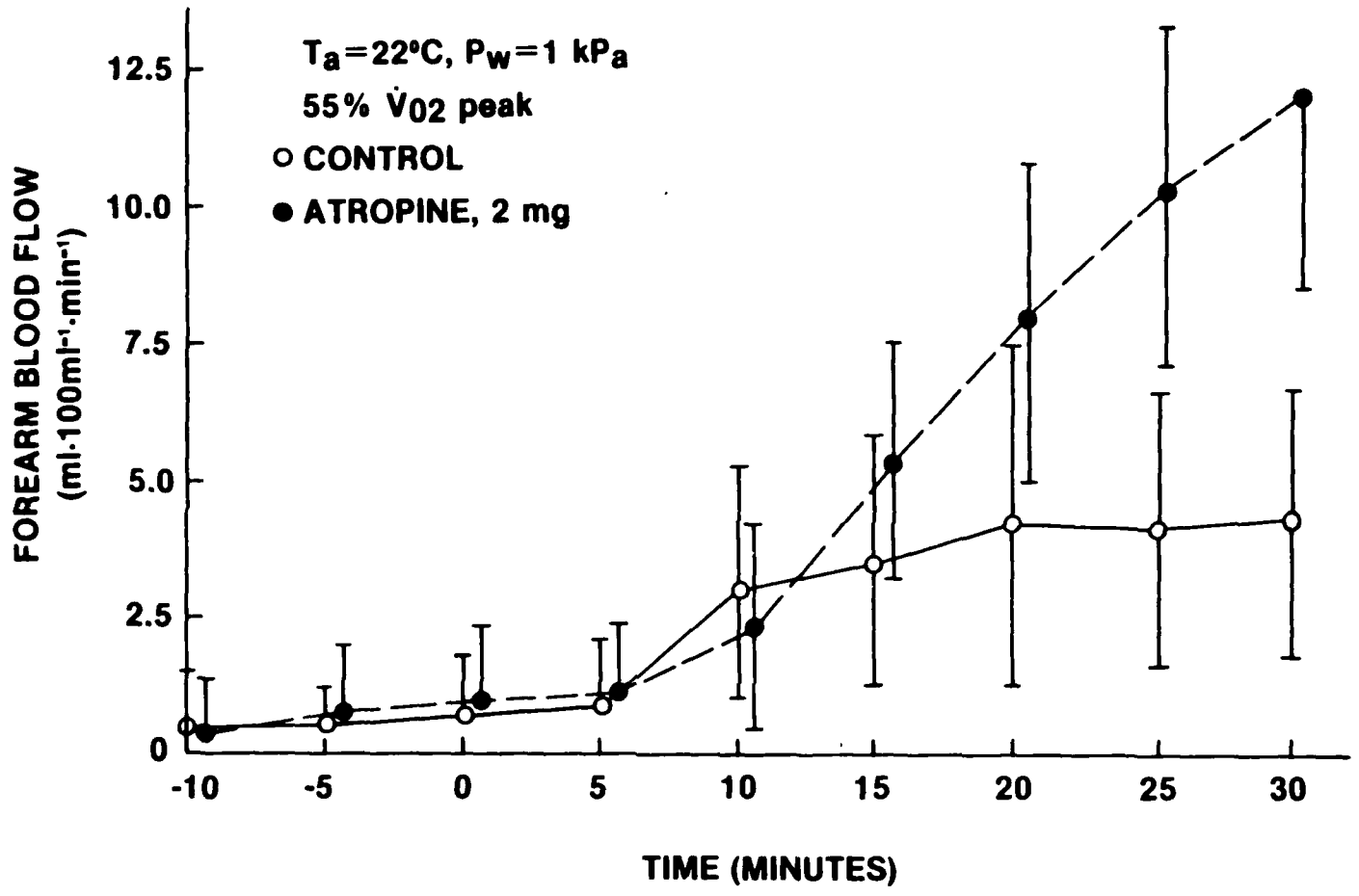
E_{max}, maximal evaporative power of the environment; E_{sk}, evaporation from the skin surface; R+C, dry heat exchange; M_{sk}, net heat flow through the skin; $P_{s,sk}$, saturated vapor pressure at skin temperature; w, skin wettedness.

FIGURE LEGENDS

Figure 1. Mean (± SD) esophageal temperature versus time in saline (control) and atropine experiments at 22°C. Exercise began at time = 0.

Figure 2. Mean (± SD) forearm blood flow versus time in saline (control) and atropine experiments at 22°C. Exercise began at time = 0.


38.0
37.0
36.0
ESOPHAGEAL TEMPERATURE (°C)
T_a=22°C, P_w=1 kPa
55% $\dot{V}_{O2}$ peak
CONTROL
ATROPINE, 2 mg
-10
-5
0
5
10
15
20
25
30
TIME (MINUTES)


T_a=22°C, P_w=1 kPa
55% $\dot{V}_{O2}$ peak
○ CONTROL
● ATROPINE, 2 mg
FOREARM BLOOD FLOW
(ml·100ml⁻¹·min⁻¹)
12.5
10.0
7.5
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2.5
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-10
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